

Durham Research Online

Deposited in DRO:

22 January 2010

Version of attached file:

Published Version

Peer-review status of attached file:

Peer-reviewed

Citation for published item:

Roberts, C. A. and Manchester, K. (2005) 'Archaeology of disease.', Stroud: Sutton Publishing.

Further information on publisher's website:

<http://www.thehistorypress.co.uk/products/The-Archaeology-of-Disease-Third-Edition.aspx>

Publisher's copyright statement:

Additional information:

Full-text of Chapter 1 deposited, 'The study of paleopathology. Paperback version published 2010.

Use policy

The full-text may be used and/or reproduced, and given to third parties in any format or medium, without prior permission or charge, for personal research or study, educational, or not-for-profit purposes provided that:

- a full bibliographic reference is made to the original source
- a [link](#) is made to the metadata record in DRO
- the full-text is not changed in any way

The full-text must not be sold in any format or medium without the formal permission of the copyright holders.

Please consult the [full DRO policy](#) for further details.

Durham Research Online

Deposited in DRO:

22 January 2010

Peer-review status:

Peer-reviewed

Publication status:

Published version

Citation for published item:

Roberts, C. A. (2005) 'The study of paleopathology', in *Archaeology of disease.*, Stroud: Sutton Publishing, pp.1-21.

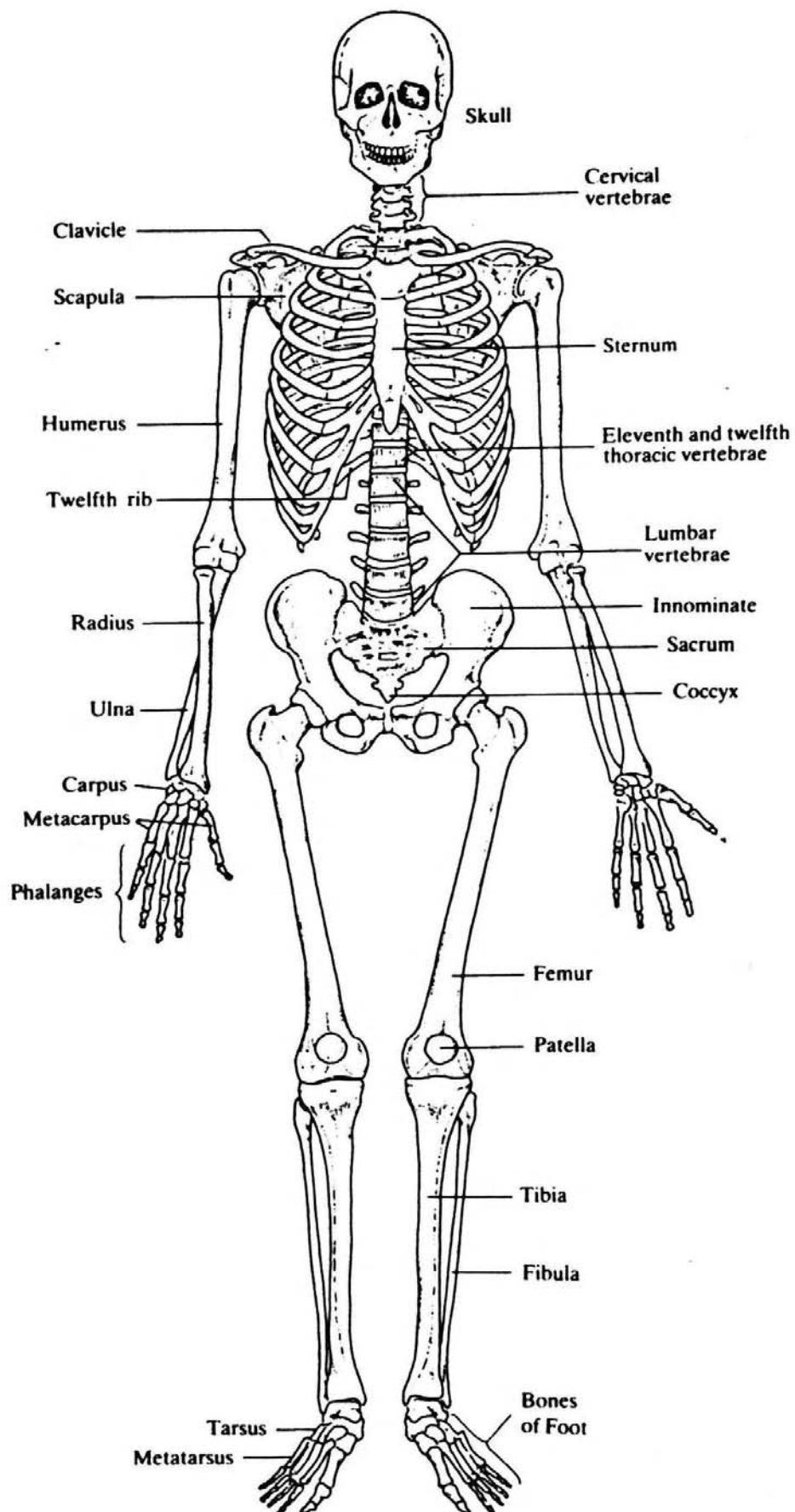
Use policy

The full-text may be used and/or reproduced, and given to third parties in any format or medium, without prior permission or charge, for personal research or study, educational, or not-for-profit purposes provided that :

- a full bibliographic reference is made to the original source
- a link is made to the metadata record in DRO
- the full-text is not changed in any way

The full-text must not be sold in any format or medium without the formal permission of the copyright holders.

Please consult the full [DRO policy](#) for further details.



The human skeleton.

CHAPTER 1

The Study of Palaeopathology

Disease is an inevitable part of life, and coping with disease is a universal aspect of the human experience . . . the experience of disease . . . is as inescapable as death itself. (Brown *et al.*, 1996: 183)

INTRODUCTION AND DEFINITIONS

The study of palaeopathology examines the evolution and progress of disease through long periods of time and looks at how humans adapted to changes in their environment. It provides primary evidence for the state of health of our ancestors and, combining biological and cultural data (the 'biocultural approach'), palaeopathology has become a wide-ranging holistic discipline. Current developments, and the future of palaeopathology, are exciting and are discussed further in the final chapter of this book.

Pathology is the study (*logos*) of suffering (*pathos*). In practice, pathology is defined as the scientific study of disease processes. Palaeopathology was defined in 1910 by Sir Marc Armand Ruffer (Aufderheide and Rodríguez-Martín, 1998) as the science of diseases whose existence can be demonstrated on the basis of human and animal remains from ancient times. Palaeopathology can be considered a subdiscipline of biological anthropology and focuses on abnormal variation in human remains from archaeological sites. The study of palaeopathology is multidisciplinary in approach and concentrates on primary and secondary sources of evidence. Primary evidence derives from skeletons or mummified remains. This type of evidence is the only reliable indication that a once-living person suffered from a health problem; whether a specific diagnosis can be made is more of a challenge. However, as Horden (2000: 208) indicates, palaeopathology 'would seem to provide our . . . hardest evidence for past afflictions'. Secondary forms of evidence include documentary and iconographic (art form) data contemporary with the time period under investigation. Unfortunately, artists and authors in the past have tended to illustrate and describe the more visual and dramatic diseases and ignored those which may have been more commonplace; the mundane, common illnesses and injuries are lost to the palaeopathologist if this type of evidence is considered alone. For example, the

mutilating deformities of the infection leprosy, the devastating effects of the Black Death, and the curiosity factor in dwarfism have led to abundant representations of these conditions in art, but coughs, colds, influenza and gastrointestinal upsets, along with cuts, bruises, burns and sprains, would probably have been so common that they would have been 'irrelevant' in the eyes of the writer or artist. In antiquity, those diseases with the greatest impact in terms of mortality, personal disfigurement or social and economic disruption probably evoked the greatest response from society (and its authors and artists). In the past, attitudes towards illness have often been due to the failure in understanding the nature of the disease itself. However, when interpreting disease in the past from secondary sources care must be taken – opinions and preferences about what should be described and drawn will affect what is read and seen. Imprecise and incomplete representation may transmit incorrect information. All literary works must be studied carefully within the traditional framework in which their facts are presented (Roberts, 1971). Those aspects of an illness which we consider to be of vital importance in the understanding of a disease may have been considered of no consequence to the observer in the past and may not therefore have been given due prominence in the record. There are also circumstances where a disease description does not correspond with any known disease in the modern world. This may be because it actually does not exist or the disease is just not recognized because of the inaccuracy of its representation. Relevant too is the need to appreciate that different diseases may produce similar signs and symptoms. For example, how does one differentiate between the skin rash of chickenpox, leprosy and measles? It is true to say that specific areas of the body may be affected by the different conditions, and the nature of the 'lesion' may differ, but to be able to determine what disease is being displayed in writing or art necessitates a very detailed representation. Another example is the clinical picture associated with respiratory disease. Cancer, chronic bronchitis and tuberculosis can all result in coughing up blood (haemoptysis) and shortness of breath (dyspnoea), but how would they be distinguished from one another in the written record if only haemoptysis and dyspnoea were being described? However, the diseases which are not displayed in the skeletal record, i.e. those affecting only the soft tissue (e.g. malaria, childhood diseases such as whooping cough and mumps, cholera and typhoid), may be recorded only in art and documentary sources, and therefore, in these cases, this type of evidence is especially invaluable. We do recognize that solely considering skeletal remains for the evidence of disease allows us to deal with only a very small percentage of the disease load in a population. However, as Horden (2000: 208) states: 'the greater the number and variety of perspectives on the pathological past with which we can engage, the greater the chance that our analysis will not be completely disabled by problems of retrospective diagnosis.'

The study of human remains within their cultural context, i.e. the period of time, geographic area and material culture, aids enormously in the interpretation of the history of disease. For example, precise dating of skeletons with bone changes consistent with venereal syphilis is important for the discussion of the pre- or post-Columbian nature and origin for this disease (Baker and Armelagos, 1988; Dutour *et al.*, 1994). Some researchers also study populations in geographic areas which sustain

contemporary traditional societies (e.g. Merbs, 1983; see McElroy and Townsend, 1996 on medical anthropology). Medical anthropology has been likened to palaeopathology because it considers disease within the population's context of living environment, diet, economy, work, etc. For palaeopathologists it is useful to interpret the archaeological (dead) population in the context of the living group if it is accepted that the latter bears close resemblances, in terms of culture, to the dead population. Of course, there are many limitations to this type of study, not least the vast differences in time and space between the living and dead populations in many cases. However, these societies are often unaffected by change (in the modern western sense) and their health and the effect of disease on their bodies is 'natural' and not influenced or changed by drug therapy. They can be, thus, useful analogues although very few societies today are immune to 'alien influences'. Nevertheless, appreciating how 'traditional' groups of people today perceive an illness, its causes and how it may be prevented undoubtedly broadens our horizons when we try to understand the impact of disease on past populations (for example, see Roberts and Buikstra, 2003).

HISTORY OF STUDY

Aufderheide and Rodríguez-Martín (1998) categorize the history of the development of palaeopathology into four phases: Antecedent (Renaissance to mid-nineteenth century), Genesis (mid-nineteenth century to First World War), Interbellum Consolidation Phase (1913–45) and New Palaeopathology (1946 to present). In the first phase work concentrated mainly on prehistoric animals (e.g. by the German naturalist Johann Friederich Esper), but there was a recognition that studying human disease would be beneficial to exploring the history of past human populations. At the end of this period the first application of the microscope to examining Egyptian mummified tissue is noted, but there was 'little scientific precision and . . . specimens (were viewed) as curiosities, not as sources of medical, pathological or historical knowledge' (Aufderheide and Rodríguez-Martín, 1998: 3). The second phase had much more of an anthropological focus, and large skeletal collections were available for study. As Aufderheide and Rodríguez-Martín (1998) point out, although 'racial' studies were the norm, pathological conditions in these collections were noted, especially by the German physician Rudolf Virchow (1821–1902). Again, it was mainly case studies that were reported and there was little consideration of what the occurrence of disease meant in epidemiological terms. Although cases provide information on, for example, the first occurrence of a disease, they are limited in providing broader views on the history of disease. Jarcho (1966: 5) also notes that researchers were so obsessed with crania they assumed 'that some diseases ended in the foramen magnum'. Happily, the study of palaeopathology today is such that students do now know that the whole of the skeleton (or as complete as possible) needs to be considered in disease diagnosis. However, as Buikstra and Cook note (1980: 435): 'we learn[t] little about population dynamics or disease evolution' from case studies. Focusing on individual experience of disease in both modern and ancient contexts can quickly lead to biased and 'patient'-centred data that may not represent the population experience from which that person derived. The French were instrumental from the late nineteenth century in developing the discipline of palaeopathology (e.g. Paul Broca, 1824–80,

who published work particularly on the evidence for Peruvian trepanation (Buikstra and Cook, 1980)). At this time, too, the first palaeopathology manual was published in America in 1886 by William Whitney.

In the third phase palaeopathology expanded and methods beyond visual (macroscopic) examination were used more often to investigate pathological lesions and improve diagnosis, in addition to statistical analysis (Buikstra and Cook, 1980). This is described as the evolution of palaeopathology as a scientific discipline. Sir Marc Armand Ruffer (1858–1917) promoted the term 'palaeopathology' as defining the scientific study of disease observed in human and animal remains. A trained physician and Professor of Medicine in Cairo, Egypt, he made detailed records of his observations particularly on mummified remains (e.g. Ruffer, 1913 in Aufderheide and Rodríguez-Martín, 1998), although, as Aufderheide and Rodríguez-Martín (1998) note, the interest in mummies then waned. Other work in Egypt came from the enormous efforts of Grafton Elliot-Smith and Frederic Wood Jones (1910; Waldron, 2000), both trained physicians, and in the early twentieth century Roy Lee Moodie in North America published two very influential books on palaeopathology (Moodie, 1923a and b, cited in Aufderheide and Rodríguez-Martín, 1998). Aleš Hrdlička was also instrumental in the development of palaeopathological studies in the Americas (1941). Located at the Smithsonian Institution (National Museum of Natural History), he created a Division of Anthropology there and accumulated large skeletal collections from North and South America for study. In tandem, Earnest Hooton of Harvard University introduced a demographic perspective to palaeopathology and used an ecological and cultural approach (and statistical analysis) to understand the disease load in the Pecos Pueblo population (1930, in Aufderheide and Rodríguez-Martín, 1998). He also advocated the accumulation of pathological specimens with known histories as a tool for comparison with the past. Aufderheide and Rodríguez-Martín (1998: 7) note that this third phase is characterized by the 'introduction and gradual standardization both of new methods and of new interpretive concepts, resulting in the emergence of palaeopathology as a scientific discipline'.

The final phase is marked by an increased recognition of the link between palaeopathology and epidemiology and demography (Aufderheide and Rodríguez-Martín, 1998), with much more of a focus on raising hypotheses and testing them with skeletal data from large numbers of individuals. Wood *et al.* (1992: 344) also note that in the 1980s and early 1990s there was a move away from 'a particularistic concern with individual lesions or skeletons to a population-based perspective on disease processes'. Notable figures in the exploration of specific diseases early in the second half of the twentieth century included Møller-Christensen (1967) on leprosy and Hackett (1963) on the treponematoses. There has also been a focus on developing standardised methods for collecting palaeopathological data (Ortner, 1991, 1994; Lovell, 2000). Additionally, the use of biomolecular methods of analysis to identify diseases, primarily the extraction, amplification and analysis of ancient DNA specific to pathogens, has seen a considerable increase in use since the early 1990s.

The Paleopathology Club, later the Paleopathology Association, was formed in 1973 and the first meeting was held in 1974 (and the first European meeting a year later in London). This still thriving Association of several hundred members

worldwide brings together people interested in, and studying, palaeopathology from a wide range of disciplines including anthropology, archaeology, medical history, medicine, pathology, genetics, biology and many more. Additionally, the World Committee on Mummy Studies, formed in 1992 after the first World Mummy Congress, 'looks after' the interests of people researching mummies, although the Paleopathology Association encompasses many of the same members. A survey of the membership of the American Association of Physical Anthropologists shows that palaeopathology as a field of physical (or biological) anthropology remains a prominent area for Ph.D. study, although not as popular as human evolution and human biological variation. It also showed that the majority of people practising palaeopathology were female, a feature that increased from the 1970s into the 1990s (Turner, 2002).

In Britain some key people in the development of palaeopathology as a discipline have included Calvin Wells (1964a), Don Brothwell and Andrew Sandison (1967), Juliet Rogers (Rogers and Waldron, 1995), Simon Hillson (1986, 1996), Theya Molleson (Molleson and Cox, 1993) and Tony Waldron (1994). However, as Mays (1997) notes, when comparing the publication content of US and UK researchers in palaeopathology, the emphasis in the UK is on 'case studies of health', whereas in the United States it is on 'population' health. In order that palaeopathology advances as a recognized discipline, the UK needs to turn more to this population approach to palaeopathology. North America, being a larger country with more research in palaeopathology being undertaken, has also seen a much longer history of study. Notable researchers here include: J. Lawrence Angel (1966a), George Armelagos (1990), Arthur Aufderheide (Aufderheide and Rodríguez-Martin, 1998), Jane Buikstra (1981), Della Cook (1994), Alan Goodman (Goodman *et al.*, 1988), Anne Grauer (1993), Robert Jurmain (1999), Clark Larsen (1997), John Lukacs (1989), Charles Merbs (1983), Don Ortner (Ortner, 2003), Doug Owsley (1994), Mary Powell (1988), Doug Ubelaker (1989) and Phil Walker (1997). This list is of course not all-inclusive but is meant to show the main publishers of work in the field.

WORKING FROM A CLINICAL BASE

The study of palaeopathology naturally starts with understanding how disease affects the body in the modern clinical sense and, more specifically, the skeleton, since most of the human-derived material palaeopathologists work with is skeletonized. It is only after this stage that this knowledge can be applied to an archaeological context. However, this process is not quite so straightforward as we might hope. For example, the classic appearance and distribution of rheumatoid arthritis in the skeleton, described in clinical texts, may not always 'fit' what we may see in an archaeologically derived skeleton. Some features may be the same, but there may be differences; however, this does not mean 'our skeleton' did not have rheumatoid arthritis. There is certainly an assumption (not necessarily correct) that the bone changes have not altered during the evolution of the disease, but we cannot be certain. Additionally, there may be skeletal changes associated with a disease in the past that are not described clinically. We must also be aware that there may have been less virulent forms of a disease in the past

compared to the present (or vice versa) that would have affected the eventual impact on the skeleton. Finally, while very subtle bone changes may be associated with disease in a living person, radiographic techniques may not identify these changes, and therefore they would not be described; in an archaeologically derived skeleton we see the bone changes but some may be puzzling when we do not see them described clinically. There are certainly some advantages to studying dry bones. But why should palaeopathology be studied?

The discipline provides a tool for investigating how people interacted with their environment and adapted to it over many thousands of years. Conversely, in modern studies of disease in living people, a doctor may only be considering a patient's progress over a few weeks, months or years. Thus, very detailed knowledge may be gained of a patient's (or group of patients') experience of a disease and the underlying reasons for its appearance. However, by considering longer periods of time we might explore major alterations in disease patterning which could have been influenced by climate and environmental change, or by significant changes in economy, housing and occupation. The disease processes studied in palaeopathology reflect the condition as seen on the skeleton or soft tissues without any influence from drug therapy, or the chronic form of the disease. What is observed is the record of a person's dental and skeletal health at the time of death. While some disease manifestations may be recognized as 'active' at death (and possibly an indicator of cause of death), most represent health insults over the period of the person's life. However, rarely can age at first occurrence of a disease be identified, because the changes observed are usually chronic, healed and long-standing. It is also possible that some disease processes today may not have been present in the past and, likewise, some pathological processes may have been present in the past but not seen today. For example, rheumatoid arthritis is a common condition today but in the archaeological record there are few convincing examples (Kilgore, 1989; Waldron and Rogers, 1994). There may be several reasons for its absence: non-diagnosis due to non-recognition, confusion with another joint disease or the fact that it really was rare in the past. It is a disease whose aetiology (cause) is ill understood. Climate, diet and environment may all have their part to play but may not, because they were different in the past, have predisposed populations to the disease.

Palaeopathology may also contribute to knowledge in modern medicine. For example, Møller-Christensen's work in the 1950s and 1960s on the skeletons buried in Medieval Danish leprosy hospital cemeteries highlighted a number of bone lesions characteristic of leprosy which had not been recognized by clinical leprologists at that time (Møller-Christensen, 1953); this work helped to identify skeletal changes of leprosy in living leprosy sufferers. A second example can be illustrated in a study by Rogers *et al.* (1990) where a palaeopathologist's and a radiologist's observations were compared. The bone changes of joint disease were recorded for twenty-four knee joints macroscopically by the palaeopathologist and radiographically by the radiologist. The results showed that subtle bone changes were not observed by the radiologist but the palaeopathologist could, on the basis of her findings, diagnose the early stages of osteoarthritis. This study was instructive in that it may explain why people today who suffer joint pain do not show radiographic osteoarthritic changes.

METHODS OF STUDY AND TISSUE CHANGE

The methods of study in palaeopathology range quite widely but usually, primarily, rely on macroscopic or visual observation and description of abnormal changes seen in skeletal remains. A description of these changes and their distribution in the skeleton or soft tissues is a prerequisite to attempting a diagnosis of the disease process being observed although, as Waldron (1994: Table 3.2) points out, diagnosis in modern contexts is difficult even with the array of diagnostic tests available. Some attempts at developing new methods of diagnosis are being explored currently (Byers and Roberts, 2003). In our description it is important to use unambiguous terminology so that readers and future workers who may wish to use these data understand its meaning, especially if they are to reinterpret the data, which may lead to a different disease diagnosis. Unfortunately, the clinical and palaeopathological literature abounds with terms describing different changes in disease, and, unless a common set of terms is used and agreed upon, there can be little hope of comparative studies of palaeopathological data on a global perspective. Buikstra and Ubelaker (1994) have gone some way towards addressing methodological standardization in palaeopathology, the British Association of Biological Anthropologists also has a similar document for use on British-derived skeletal material (Brickley and McKinley, 2004), and the 'Health in Europe Project' overseen by Richard Steckel, Clark Larsen and Philip Walker also aims to standardize the recording of thousands of skeletons so that comparative research can be undertaken.

The bone changes seen in palaeopathology usually represent chronicity, i.e. the individual adapted to the problem and the body reacted to it by forming and/or destroying bone. These people survived the acute phase of the disease and progressed into the chronic stage. An individual with skeletal abnormalities may therefore represent a healthier constitution than one without, although lack of any bone abnormality could either mean a healthy individual who died as a result of an accident, for example, or somebody who was unhealthy but died before bone change occurred; absence of evidence does not mean evidence of absence in all cases! In addition, Wood *et al.* (1992: 357) suggest that 'different disease processes interact with each other and also with an individual's constitutional susceptibility to stress in determining frailty', and hence what is observed on the skeleton. However, the degree of frailty in a population is not known for the past, nor is its association with the development of abnormal lesions, and knowledge of the amount and length of exposure a person had to a disease-causing organism is limited.

The bone changes of disease may be proliferative, i.e. bone forming and initiated by osteoblasts (bone-forming cells), or destructive, i.e. bone destroying and initiated by osteoclasts (bone-destroying cells). There may also be a mixture of the two activities. In the normal physiological state there is a balance between osteoblast and osteoclast activity which allows continuous remodelling and turnover of bone throughout life. However, as a person ages, bone loss overtakes bone formation and there is net loss of bone. Pathological stimuli may induce an imbalance, producing changes of atrophy, hypertrophy, hyperplasia or metaplasia. The cellular changes in bone are stimulated by a change in oxygen supply to the tissues – high blood oxygen tension stimulates osteoclast activity and low blood oxygen tension stimulates osteoblasts. Hypertrophy involves increase in cellular

size and may be induced physiologically, e.g. the person has a heavy manual occupation and the muscles used become increased in size. Atrophy means that there is a decrease in cell size, e.g. when a limb is not being used in, say, paralysis of whatever cause. Hyperplasia indicates cellular division and an increase in cellular content of the tissue, and metaplasia involves the change in differentiation of cell type, i.e. a cell assumes the morphological and functional characteristics of another cell under pathological stimulus, e.g. in a tumour.

The bone formed in a disease process may be woven (or fibre), immature or primary bone (porous, disorganized; Fig. 1.1), or more mature, older, organized, lamellar bone (Fig. 1.2). The former indicates that the disease process was active at the time of death, and the latter indicates that the process was quiescent or had been overcome. However, the presence of active lesions may not indicate the process was the cause of death but that, with other factors, it contributed. It is also of importance to study whether an abnormal lesion appears healed (smooth bone with rounded edges) or unhealed (sharp unremodelled edges) because this gives an indication of the disease state at the time of death and perhaps whether this abnormality had contributed to the demise of the individual. However, determining the ante- or post-mortem nature of unhealed lesions can prove problematic.

It is essential to have a complete skeleton to study since observation of distribution patterns of abnormalities is necessary to attempt a diagnosis based on modern clinical criteria. Unfortunately, in archaeological contexts complete skeletons are not usually the normal occurrence and the palaeopathologist is often working with incomplete data. It should also be remembered that several diseases may induce similar lesions on bone and can occur on the skeleton at the same time, because bone can only react to a pathological stimulus in a limited number of ways, as we have seen. For example, new bone formation on the lower leg bones (tibia and fibula) may represent leprosy, treponemal disease, tuberculosis, trauma,



Fig. 1.1. Long bone with woven bone formation on top of the original cortex.



Fig. 1.2. Long bone with lamellar bone formation.

non-specific infection and scurvy. Of course, one would never diagnose any of these conditions solely on the basis of this change, because we would be considering the fuller picture (distribution pattern) of all the changes. Consideration of possible differential diagnoses for the abnormalities described is essential because of the potential for several disease processes to cause the same bony changes. This means recording the bone abnormalities and their distribution and considering all potential disease processes which could have caused the patterning; by a process of gradual elimination on the basis of known patterning in modern clinical circumstances a most likely diagnosis may be made. However, it may not be possible to make a definite diagnosis. Some workers in the field also like to attach some degree of 'severity' to lesions observed, but their appearance may not necessarily reflect a gradation in the disease. If grades are to be included, a definition of the grades (including photographs) should be given so that future researchers understand the meaning of the definitions. Recording detailed descriptions of abnormal changes, although accepted as essential, does take up space in a skeletal report but may be solved using CDs, microfiche or web archives. However, it is advocated that an archive is kept for all reports. Advances in the storage of both visual and textual data since the 1990s, may help this problem to be solved in the future. The use of zip and compact disks for the recording of large amounts of data has allowed the transmission of these data to other readers. Additionally, electronic transmission of images captured by digital cameras, and of scanned photographs, has enabled researchers to gain opinions on pathological specimens and their diagnosis much more quickly than previously. We also have wide access to the worldwide web, where web pages record type specimens of specific diseases, and the information can be accessed by anybody with the technology to do so.

Of especial interest to the palaeopathologist is the study of disease prevalence through time but basic data must be collected before meaningful prevalence rates can be obtained (see Waldron, 1994 for a discussion of the definitions of prevalence and incidence and their relevance to past human skeletal populations). For example, if the prevalence of left hip joint disease is to be studied, then the observers need to know how many left femurs and acetabulae they have observed in order to determine the prevalence of joint disease of the component parts of the hip – inventories of bones and teeth observed are essential data which should be included in all reports (Table 1.1).

Table 1.1 Prevalence of left hip joint disease in three hypothetical skeletal populations

| <i>No. of acetabulae</i> | <i>No. affected (%)</i> | <i>No. of femur heads</i> | <i>No. affected (%)</i> |
|--------------------------|-------------------------|---------------------------|-------------------------|
| 20 | 10 (50) | 30 | 10 (33.3) |
| 55 | 20 (36.4) | 75 | 15 (20) |
| 130 | 60 (46.2) | 115 | 33 (28.7) |

Note: One often does assume that if one bone of the joint is affected then the apposing element will similarly be affected, but this is not always the case. However, in the above example the frequency of joint disease in both acetabulae and femur heads in individuals with both elements surviving should also be examined.

The nature of the (often) fragmentary state of human skeletal material means that one cannot assume all bones are represented in all skeletons and, if prevalence rates for disease are presented according to individuals, e.g. five out of ten people had leprosy, the assumption has to be that all bones (facial, hand, foot and lower legs) were present for observation (even though five of the unaffected skeletons may have had no foot bones to observe).

In addition to macroscopic examination of the skeleton, radiography (Fig. 1.3) plays a large part in the diagnosis of disease and trauma (Roberts, 1989; Blondiaux *et al.*, 1994; Hughes *et al.*, 1996), especially in the case of unwrapped mummies (Zimmerman, 2000). Light, transmission and scanning electron microscopy (Martin, 1991; Bell and Piper, 2000; Pfeiffer, 2000) add an extra dimension and can increase accuracy for diagnosing disease (Fig. 1.4) and also pseudopathological changes, i.e. those post-mortem changes which appear to be pathological but are not. Physical and chemical techniques of analysis have been used increasingly over time to diagnose disease (e.g. lead poisoning, Vuorinen *et al.*, 1990; Klepinger, 1992) and also to examine dietary status (Katzenberg *et al.*, 1996; Wright and Schwarcz, 1998; Katzenberg, 2000; Lillie and Richards, 2000; Sealy, 2000; Cox *et al.*, 2001; Dupras *et al.*, 2001); of course, the latter has a bearing on a person's likelihood of acquiring a disease. More recently, work has focused on identifying disease at the molecular level, and there have been considerable advances in this area since the second edition of this book (e.g. Salo *et al.*, 1994; Brown, 2000; Gernaey and Minnikin 2000; Stone 2000; Taylor *et al.*, 2000).

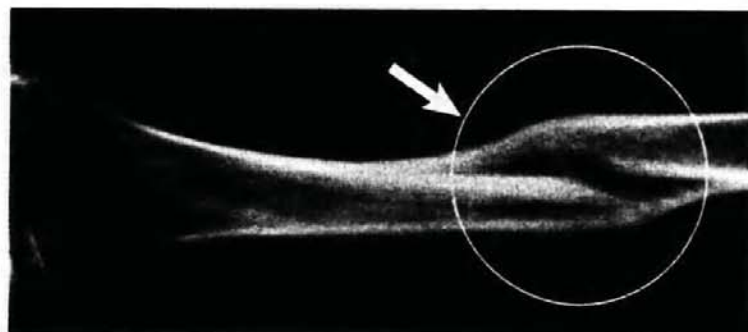


Fig. 1.3. Radiograph of tibia showing healed fracture.

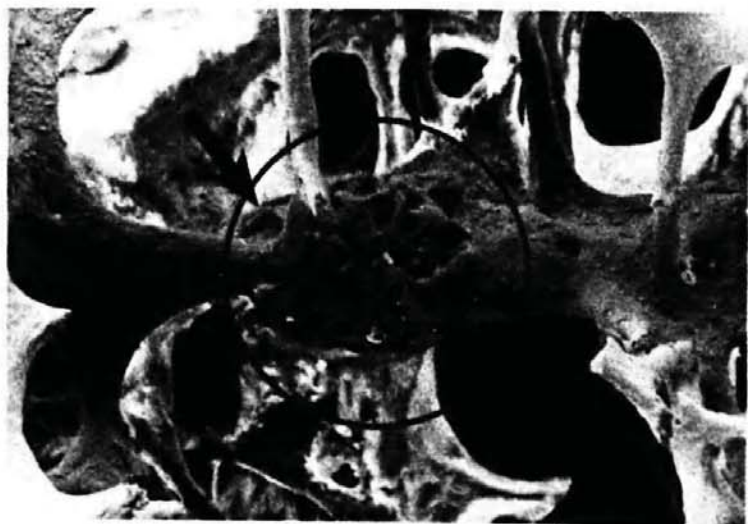


Fig. 1.4. Scanning electron microscopy of section of a lumbar vertebral body showing a healed microfracture (early Medieval, eighth–tenth centuries AD, Raunds, Northamptonshire, England).

Since the 1990s attempts have been made to suggest how abnormalities should be recorded and to specify the minimum set of data which should be generated for skeletal population studies (Rose *et al.*, 1991; Buikstra and Ubelaker, 1994; Brickley and McKinley, 2004). Additionally, experimental studies have shown that there can be quite marked discrepancies in how data are recorded (Waldron and Rogers 1991; Miller *et al.*, 1996). To be able to compare data between different cemetery groups, methods of recording and the data generated must be comparable if palaeopathology is to be recognized as a scientific discipline.

TERMINOLOGY

There are several terms that the reader should become familiar with. **Aetiology** refers to the cause of the disease, **pathogen** is the foreign life-form which is capable of stimulating disease (e.g. *Mycobacterium tuberculosis* causes tuberculosis), and **pathogenesis** refers to the mechanism and development of tissue change in a disease. An affected individual's physical **signs** and **symptoms** are **clinical features** (e.g. the swelling and pain of joint disease respectively), and a **lesion** refers to the individual tissue manifestations in a specific disease. **Epidemiology** studies the incidence (or prevalence), distribution and determinants of diseases in populations. For example, pollution in an environment may determine the prevalence of upper respiratory tract infections. **Mortality** refers to death and **morbidity** describes the occurrence of illness. Clearly, there may be many factors contributing to the occurrence of disease – genetic predisposition, age, sex, ethnic group, physiological state and social status, prior exposure to the micro-organism, intercurrent or pre-existing disease and human behaviour, e.g. occupation, diet, hygiene (e.g. see Polednak, 1989 on racial and ethnic differences in disease, McElroy and Townsend, 1996 on ecological factors). A person may also have natural (i.e. inherited) **immunity** to a disease independent of any previous exposure to specific pathogenic micro-organisms. In addition, an acquired adaptive immunity may be stimulated by exposure to foreign proteins of invading pathogenic micro-organisms and the immune system will be dependent upon the properties of specific circulating white blood cells called lymphocytes. Adaptive immunity is characterized by the retention of a specific memory for the invading pathogen so that a 'tailor-made' defence mechanism for future invasion by the specific pathogen is in place. The problem with immunity in past human groups is that the levels of natural and acquired immunity cannot be ascertained. However, chronic evidence for disease does indicate that a person's immunity was effective enough to prevent death in the acute phase. A child who died with no bone changes of disease may also indicate that his or her immune status was not developed enough to prevent disease. Another example would be that a person with bone changes of leprosy usually has the lepromatous (or low-resistant) form of the disease, indicating a less-developed immune system (Fig. 1.5). As time goes by people may 'move' their immunity to the other end of the spectrum and develop tuberculoid leprosy, because of increased exposure (and adaptation) to the infection. Clearly, building up one's immune system by being exposed to pathogens in the environment is key to a healthy life (Hamilton, 1998).

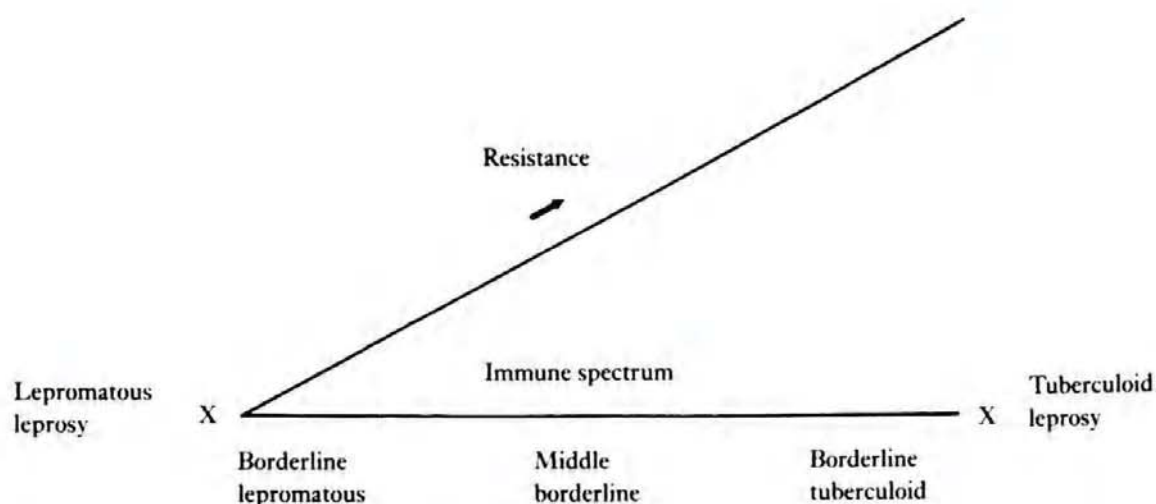


Fig. 1.5. Immune spectrum of leprosy; those with lepromatous leprosy are more easily identifiable in the skeletal record. (After Ridley and Jopling, 1966)

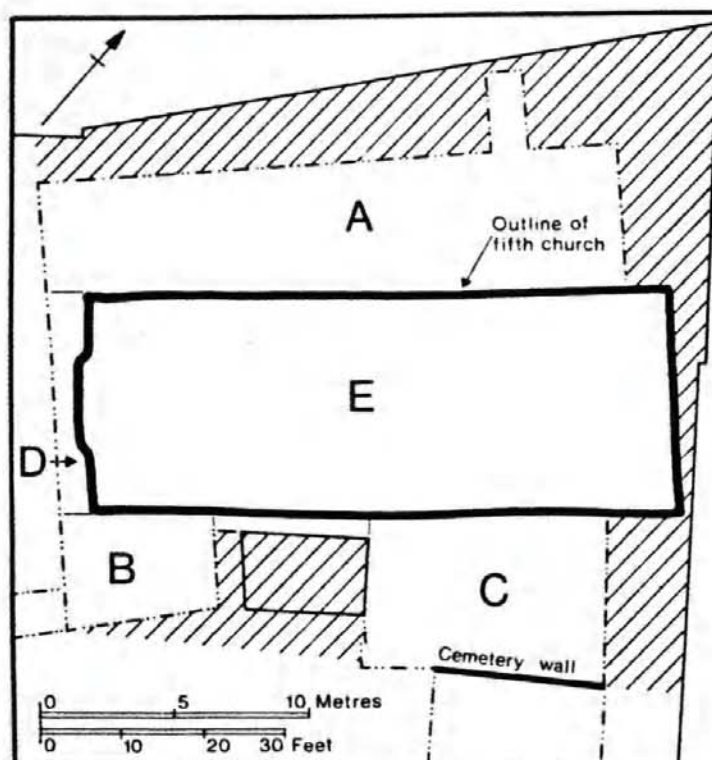
LIMITATIONS OF PALAEOPATHOLOGICAL STUDY

There are several limitations to the study of palaeopathology, as Wood *et al.* (1992) stated. In any discipline there are limitations, but some can be overcome. The hazards of selective mortality, individual variation in a person's risk of disease and death (i.e. there is an unknown mix of individuals who varied in susceptibility to death and disease, depending on biocultural factors), and the non-stationary nature of populations were highlighted by Wood *et al.* (1992) as major problems which it may not be possible to solve in palaeopathology. The following summarizes other limitations that should be considered.

The 'populations' being studied in palaeopathology are dead and therefore may not be representative of the living group; biological anthropologists are dealing with a sample of a sample of a sample . . . of the original living population, and total excavation of a cemetery is unusual. Partial excavation of a cemetery is the most common occurrence in archaeology and therefore only a portion of the original buried population will be examined (Fig. 1.6); the differential disposal of males, females, children and people with particular diseases, and their subsequent excavation, means biases in the produced data are inevitable. For example, in some cultures children were not always buried in the cemetery serving the general population – for example, in the Roman period in Britain (Philpott, 1991). In addition, skeletal material is often fragmentary and poorly preserved, with non-adult skeletons commonly suffering post-mortem damage (see Guy *et al.*, 1997), and therefore observation of the distribution pattern of abnormal changes is not possible; hence an attempt at a diagnosis often cannot be made. Researchers in biological anthropology often deal with small numbers of individuals and therefore cannot say much about disease prevalence at the population level because the group of skeletons being examined can only be a small sample of the original living population; sample representivity is often difficult to assess.

Acute infective disease is likely to have killed people very quickly in antiquity, especially if the individual had had no previous exposure or experience of the

Fig. 1.6. Plan of the excavation of the late Medieval church (E) cemetery (twelfth–sixteenth centuries AD) of St Helen-on-the-Walls, York, England; the hatched areas are those not excavated that may contain age and sex specific groups of people that could change the demographic profile of the populations, if excavated. (From Dawes and Magilton, 1980, with permission of York Archaeological Trust)



invading organism. Therefore, no evidence of abnormal bone change would be visible (or expected) because the person died before the bone change developed. Many diseases also only affect the soft tissues and therefore would not be visible on the skeleton. It is therefore quite possible that skeletons from the younger (non-adult) members of a cemetery population were victims of an acute, or soft tissue, disease because frequently they do not have any signs of abnormal bone change. Additionally, their immune systems may not have been fully developed to defend against disease. Furthermore, pathological bones are inherently fragile structures and may, in some circumstances, become damaged while buried and not survive to be excavated, which precludes examination and recording; thus their frequency may be under-represented.

A further factor to consider is the inability, in most circumstances, to ascribe a cause of death to an individual. Without, for example, a weapon embedded in the skeleton in the grave, or an unhealed injury (Fiorato *et al.*, 2001), it is often guesswork determining a cause of death, although the observation of the posture of a skeleton within its grave may be an indication of cause of death. For example, the 'live' burials recorded from Kingsworthy, Dalton Parlours and elsewhere in Britain (Hawkes and Wells, 1975; Manchester, 1978a) were dependent for interpretation upon the observed posture. Beheadings, seen as cut marks to the neck vertebrae (Boylston *et al.*, 2000), or hanging, strangulation or trauma to the neck, seen in fractures to the hyoid bone or ossified neck cartilages, may also be clues. However, complete bodies such as those from north-west European bogs (Brothwell, 1986) may indicate a more obvious cause of death because of the survival of soft tissue. What can be indicated are the disease processes an individual may have been suffering from in life and whether the disease was active or not at the time of



Fig. 1.7. Post-mortem (pseudopathological?) lesions around the eye socket due to gnawing from a rodent in the grave. (*Calvin Wells Photographic Collection*)

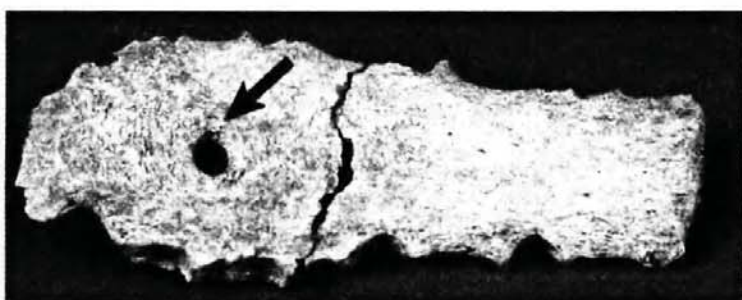


Fig. 1.8. Sternal foramen, a non-metric trait.

death. However, we should not dwell too much on our inability to assign a specific cause of death to skeletal remains. There is ample evidence from clinical research and historical data that assigning the correct cause of death was not, and is not, easy (see Hardy, 1994 on eighteenth- and nineteenth-century Cause of Death Statistics for England and Wales, and Alter and Carmichael, 1999 and Hanzlick, 1997 on the history of registration of causes of death). For example, a study of Irish general practitioners by Payne (2000) found that up to 50 per cent of cause of death data on death certificates could be based on guesswork. Likewise, Ermene and Dolene (1999), after correlating cause of death data on death certificates and autopsy reports in 444 individuals, found in 49 per cent of cases there was complete agreement, and in 19 per cent complete disagreement.

Apart from determining cause of death, there is also the problem of deciding whether abnormal bone change is the result of a disease or due to the post-mortem effects of deposition, burial and excavation of the body, or pseudopathology (Fig. 1.7 and Wells, 1967; Hackett, 1976; Bell, 1990). Finally, one should be careful of ascribing disease to an individual on the basis of normal variation in the skeleton, or non-metric trait presence (Fig. 1.8; e.g. see Saunders, 1989 regarding non-metric traits of bone and Scott and Turner, 1997 for teeth).

BIOCULTURAL PERSPECTIVES OF DISEASE FREQUENCY

Despite these limitations, a striking feature in the study of the history of disease is the constant nature and the different distribution of disease with the passage of time. Many diseases which have been recognized in skeletons from distant antiquity present the same physical characteristics as those diseases today. Diagnoses in

palaeopathology are made with reference to the knowledge of modern pathology as we have seen. The agents of disease stimulate bone reactions which we assume were the same for the palaeolithic hunter as they are for the twenty-first-century office worker. However, with the development of ancient DNA analysis very recent work has started to explore whether strains of specific diseases were the same today as they were thousands of years ago (Buikstra, pers. comm.; Zink *et al.*, 2003). Nevertheless, it is the overall world frequency of disease and the differing geographical patterns of disease which have changed during the history of human populations. The following sections consider a number of themes and their impact on health.

Movement of people

Travel, trade and contact with people have spread disease, sometimes with devastating effect, and this is still seen today.

The human infectious diseases have achieved worldwide status through the migrations of humans and the animals associated with them (Wilson, 1995). For several thousands of years armies have crossed frontiers and seas and travelled on campaign to distant lands. Crowded together, poorly nourished and usually exhausted by the stress of battle, soldiers on active service are notorious for their spread of infectious disease, often of the enteric types. Today, refugees from war-torn areas of the world often endure similar living conditions in their new environment. They hope for a better life, but this is not always achieved immediately, and they take their diseases with them as they travel, while experiencing new health insults on weakened bodies (see Roberts and Buikstra, 2003, on the effect of travel and migration on the frequency of tuberculosis).

Unlike the immunity of indigenous populations as, for example, in the tropical diseases, people transporting infectious disease from one region to another were probably overtly infected themselves. With the notable exception of typhoid fever, there are very few asymptomatic carriers of human infectious disease. The population into which the disease was introduced was also no more and no less susceptible than the people actually transporting the disease.

When one population moves from the region to which it has become adjusted, to another, it shows increased susceptibility to the diseases of the area into which it moves (Mascie-Taylor and Lasker, 1988; Roberts *et al.*, 1992). This fact was noted with cynical effect in Kent in the nineteenth century. At that time, and for many years before, the north coast of Kent was an important focus of endemic malaria. The area was marshy and the frequent hot, dry summers resulted in outbreaks of the disease (Dobson, 1994). However, indigenous males appeared to be immune to a strain of the malaria parasite and so did not readily succumb to the disease. Another example is the effect of explorers from the Old World on the native population health of the Americas: new diseases were introduced to which they had no resistance (Larsen, 1994; Larsen and Milner, 1994).

Climate and weather

The latitude, longitude and associated climate and weather have a profound effect upon the incidence of certain diseases (Brimblecombe, 1982; Patz *et al.*, 1996; and see Lukacs and Walimbe (1998) for a palaeopathological example), and the

constant relationship between respiratory disease and more recently seasonal affected disorder (SAD) and the winter climate is well known to all living in northern Europe. What may not be quite so well known is the seasonal and climatic variance of such diseases as meningitis, poliomyelitis, glaucoma and mental disease. It is possible that a knowledge of the geographical prevalence of specific diseases will provide clues to their causes (Learmonth, 1988). However, the ability of people to adapt to a totally new environment, climate and weather, and the associated diseases, is perhaps one of our most valuable characteristics.

Diet and economy

Until the advent of agriculture in all parts of the world, many people lived in reasonable harmony with their environment. The equilibrium was destroyed with deforestation and the development of farming. This still continues to be a problem (Morse, 1995). Ploughing, crop-rearing and tending flocks also increase exposure to new organisms. For example, cultivated soil containing organic refuse, particularly animal dung, is a good medium for survival of the spores of the tetanus bacillus. People cultivating land were liable to develop tetanus, which in antiquity must have been almost invariably fatal. In common with most of the acute infectious diseases, tetanus is not recognizable in the human skeletal record. We also know that some bacteria may survive for considerable amounts of time and be still viable (e.g. tuberculosis – Cosivi *et al.*, 1995). The use of dung for fuel (Fig. 1.9), building and manuring could potentially introduce health hazards.

Environmental change has been a feature of all periods of time. In association with the change in environment, be it deforestation, land cultivation or urbanization, people have come to live in closer relationship with a variety of animals. Cattle, horses, sheep and pigs were accumulated and people lived life in close proximity to them, often sharing their houses. Only later were the dog, cat and a multitude of other animals seen as companions and pets. These animals are all subject to their own parasites which may or may not cause disease within them. Cattle are subject to tuberculosis, the pig to *Taenia solium* (tapeworm) and the dog and sheep to hydatid disease, to name but a few. In fact, many of our human diseases may have originated from animals (Waldron, 1989: table 3). Increasing domestication of animals brings people closer not only to animals but also to their parasites, be they worms, bacteria or viruses, and it may have been during this time of increasing contact with animals that people first became infected with the parasites of animal origin (zoonoses – see Brothwell, 1991). Close contact with



Fig. 1.9. North-west China: large pile of animal dung used for fuel for this nomadic population.

dogs and canine distemper may have been responsible for the introduction of measles to humans. The measles virus, which at present appears to have no primate ancestral parallel, is similar to the virus causing canine distemper. This transfer may have been the stepping-stone for the recurrent endemic and at times life-threatening disease of measles with which modern populations are so familiar. However, the community size at the introduction of the measles virus must have been large enough to sustain it as an endemic infection.

In the Americas, the introduction of agriculture, particularly maize, allowed the development of a more settled community with permanent housing to enable people to care for crops and animals. However, as population numbers increased, the local living environment became less healthy, diet became less varied and people's health suffered. Studies from the Americas consistently indicate a decline in health with the advent of agriculture (Cohen and Armelagos, 1984; Cohen, 1989; Larsen, 1995; see table 1.2 for an example) and note that hunter-gatherers were probably healthier because of higher mobility, less fat intake, a varied (and more reliable) diet and temporary housing. However, this does not mean that they did not suffer. For example, disease could be transmitted from hunting, butchery and consumption of wild animals, and water sources could become polluted.

Living environment

The rise of urban communities, which gathered momentum towards the later Medieval period in Europe certainly, pushed increased numbers of people into closer contact, often in poorly ventilated, unhygienic houses, creating a situation that allowed transmission of infectious diseases more readily (Keene, 1983; Woods and Woodward, 1984; Cohen, 1989; Dyer, 1989; Rosen, 1993; Howe, 1997).

In the early and somewhat haphazard stages of village and town development, little thought was given to waste disposal (Keene, 1983). The health hazard of the open sewer and its attendant flies was not realized. The inadequacy of communal water supply was unrecognized (see Fig. 1.10 for a contemporary example). It is within this framework of public health ignorance that the largely water-borne infections of cholera, typhoid and infantile gastroenteritis flourished. These are the debilitating, sometimes fatal, illnesses of adulthood and the almost invariably fatal illnesses of infancy and childhood. The almost careless, at least unwitting, proximity of water supply and effluent discharge in the narrow Medieval



Fig. 1.10. Kathmandu, Nepal: children playing in a highly polluted river full of rubbish.

town streets of Europe allowed the easy transference of bacteria and viruses from one public service to the other. Later in time, a specific example in London reveals the problem of having a water supply which may not be beneficial to health. In 1854 the Soho area of London was subject to an epidemic of cholera and its source was centred on a pump in Broad Street (now Broadwick Street). Once the pump was removed from use, the infection declined. This suggested that the water supply had been infected, a common method of transmitting the disease (Learmonth, 1988).

In the twentieth century the health hazards of the large conurbations of industrial development have become apparent, albeit poorly understood. Lung cancer and chronic bronchitis showed a high incidence in the large centres of population in Britain (Howe, 1997). The coal-miner's pneumoconiosis and anthracosis, also seen in past humans (Munizaga *et al.*, 1975; Walker *et al.*, 1987), the business executive's coronary thrombosis due to stress, the ubiquitous mental illness, the gut and lung cancer of the developing world due to changes in diet, the adoption of smoking and the increases in health problems due to environmental pollution (Hassan *et al.*, 2003) are but a few of the many penalties of human adaptation to changing circumstances. The phenomenon is not new but is better documented today. Nevertheless, not all environmental change has favoured the parasite. Sometimes, quite unintentionally, people have altered the environment and destroyed the natural habitat of the vectors of some diseases and so, effectively, eliminated the particular disease. Drainage of marshlands and maintenance of adequate dykes were responsible for the eradication of malaria in the late nineteenth century in some parts of Britain. This environmental improvement, carried out by the farming community for reasons of economy, led unwittingly to the elimination of the mosquito by destroying the habitat favourable to it.

It is not only the change in landscape which results in disease variance; we must also consider the differences the range of environments could have on disease frequency. Coastal and inland, island and mainland, river, lake and estuary, highland and lowland, hot and cold, dry and humid; all these environments affect the range of diseases experienced. Occupation of unchanged land itself may also encourage the development of certain diseases. It has been suggested, for example, that people living in districts with a high soil content of copper, zinc and lead have a higher than average incidence of multiple sclerosis (Warren *et al.*, 1967), and copper mining in the past could lead to poisoning (Oakberg *et al.*, 2000). In Jordan, high levels of copper today affect populations' health (Pyatt and Grattan, 2001), and in the south-west of England granite-walled houses emit radon that could cause cancer. The causal relationship between the development of goitre and a nutritional deficiency of iodine is well known. This deficiency, due to a low iodine content of water, is most common in inland mountainous areas of the world, especially in parts of America and Switzerland (Drury and Howlett, 2002). In Britain the deficiency gave rise to the now classic 'Derbyshire neck'. The significance of fluorine as a nutritional trace element is a recent concept, although as early as 1892 it was suggested that a dietary deficiency of fluorine was related to the high prevalence of dental caries in Britain. Fluoridation of drinking water in Britain has caused great controversy over the years but studies do show that it reduces the frequency of caries in children (Thomas *et al.*, 1995). The properties

of fluorine at the correct levels in the prevention of dental caries are now well known. It is also known, however, that excessive levels of fluorine in water can cause fluorosis (Blau *et al.*, 2002). However, differentiating between a disease caused by a deficiency or lack of a dietary element noted in skeletal and dental remains, and the infiltration of soil elements into the bone or teeth, needs great care in interpretation (Price *et al.*, 1992). Such problems of the relationship between disease and environment are ill-understood today. Their significance for the diseases of antiquity may remain unknown. The difference today is that an association between disease incidence and 'geographical' characteristics can be assessed and checked in contemporary societies; for the past this is more difficult.

There is also a factor in the causation of disease which is beyond the influence of the environment and which may have a bearing upon the differing geographical prevalence of certain diseases of antiquity. In 1953 it was reported that there was a significant association between cancer of the stomach and individuals of blood group A (Aird and Bentall, 1953). Since that time investigation has extended to many diseases and blood group associations (Vogel, 1970; Polednak, 1989), including the relationship of disease to certain proteins of the blood (Cattaneo, 1991). The results of these investigations are not without their critics (Weiner, 1970), but, as is observed, blood group frequencies do separate geographically, even in the present days of widespread travel.

Occupation

The health hazards of the type of work people have done, and do, are clear. You may be a hunter-gatherer and live in a healthy environment with a good well-balanced diet, but the dangers of trauma from hunting wild animals may compromise your health considerably! Working in the pottery, textile and mining industries creates particles in the environment that, when inhaled, can induce inflammation and infection in the respiratory tract (e.g. Lancaster, 1990). The trades of tanning, butchery and farming create an environment conducive to contracting zoonoses (e.g. see Reber, 1999 on tuberculosis in nineteenth- and twentieth-century Argentina), and spending long hours cooking over a smoky fire (Fig. 1.11) may lead to infection and cancer of the respiratory tract (Larson and Koenig, 1994; Dietz *et*



Fig. 1.11. China: woman cooking over a smoky fire.

al., 1995). In the latter part of the twentieth century and now in the twenty-first century, certainly in westernized societies, health and safety measures have been introduced to prevent disease and injury, such as putting guards over dangerous machinery, and ensuring that people working in noisy or polluted environments wear ear-muffs and masks, respectively. However, in the past these regulations (in a less developed form) may or may not have been instigated. It is, nevertheless, likely that preventive measures were inconsistently exercised.

Treatment

The commonplace infections which killed or debilitated humans in antiquity are rapidly treated with antibiotics in modern western societies. Unfortunately, the use, and misuse, of the earlier antibiotics has led to the development of resistant strains of bacteria, for example in tuberculosis today (Grange, 1999), and in some instances the parasite has regained the upper hand. The manufacture of more and varied antibiotics has, however, once more mastered some diseases. Infectious diseases due to viruses are in a different class, since at present no universal and totally effective antiviral agent exists. The common cold, influenza, measles and smallpox, for example, are incurable once established. Success against them depends upon preventing their establishment. With very few exceptions, however, these viral diseases are not manifest in skeletal material and for this reason will not be discussed further.

More important for western populations is the increasing significance that circulatory, degenerative and neoplastic disease has in modern society. By their adaptability and knowledge, humans have exchanged one group of diseases for another. The conquest of cancer, AIDS (acquired immune deficiency syndrome) and circulatory disease remains a goal for the present. The increase in incidence of these diseases may be more apparent than real and due in part to the increased longevity of modern western populations. They are also due to environmental change and the industrialization of the past two hundred years. Of course, in the past sophisticated methods of treating illnesses, e.g. using drugs, did not exist and would not have affected the course of the disease. However, what we would now call 'alternative therapies' were clearly exploited, as seen in documentary and artistic representation. They include blood-letting, including cupping (Fig. 1.12), to rebalance the humours, cautery (the application of hot irons to the affected part), herbal remedies, minor and major surgery such as setting fractures, amputation and trepanation, wound care, bathing and more unconventional remedies (Rawcliffe, 1997). We also have records for the founding of hospitals for specific diseases such as leprosy (Roberts, 1986a) and tuberculosis (see summary of sanatoria development in Roberts and Buikstra, 2003), although whether particular treatment regimes were used are debated. In Medieval Europe hospitals were often founded by a benefactor who was usually more interested in 'getting to heaven' than in treating the sick effectively. We know too that 'medical' practitioners existed, and ranged from village elders to barber-surgeons and bone-setters. Despite this long list of 'available' care and treatment, we do not know what proportion of people through time had access to therapy, whether only higher social status (older/younger?) males or females were favoured, and whether urban or rural populations were more likely to be treated. We know today that certain parts of populations are advantaged for various reasons (e.g.

see Roberts and Buikstra, 2003 on the problems of access to treatment for tuberculosis) and it is highly likely that this was the case in the past.

The problems of disease today in relation to environmental change, to advances in medical treatment and to the very nature of humans themselves are complex and the subject of continuous change. The understanding of disease in antiquity and the analysis of the changing patterns of disease throughout history are equally complex, but may be of paramount importance in the interpretation of medical problems today. While we may not detect all our ancestors' health history, we may start to understand what the presence of some diseases meant in terms of absolute impact.

In the following chapters diseases that potentially affect bones and teeth are discussed. Both congenital and acquired diseases are considered. Congenital disease is present at birth, and acquired disease is developed during life. This latter classification encompasses:

1. Dental disease: those diseases or conditions affecting the teeth and associated tissues.
2. Traumatic lesions: due to injury or malformation of the skeleton and associated soft tissues.
3. Joint disease: diseases that affect the joints of the body and associated tissues.
4. Infectious disease: caused by invading living organisms (viruses, bacteria, parasites or fungi).
5. Metabolic disease: caused by a disturbance in the normal processes of cell metabolism.
6. Endocrine disease: caused by over- or underactivity of the endocrine glands which secrete hormones.
7. Neoplastic disease: 'new growths' which may be benign (localized to the site of growth) or malignant (progressive growth which invades and destroys surrounding tissues and spreads to more distant sites in the body).

In a book such as this it is not possible to consider all the possible skeletal and dental diseases that occur in past human remains; it is the intention to deal with those disease processes that are more commonly seen, with the aim of providing guidelines for scholars in the discipline and informing other interested readers about commonly occurring palaeopathological lesions and their interpretation within a cultural (archaeological) context.



Fig. 1.12. China: treatment by cupping (a heated glass vessel is placed on the skin; this creates a vacuum and draws the blood to the surface).